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## Chemotaxis in Myeloid-Derived Suppressor Cells: Characterizing the Interaction between TFF2 and CXCR4

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## Natural Science

### Poster

Title: Chemotaxis in Myeloid-Derived Suppressor Cells: Characterizing the Interaction between TFF2 and CXCR4

Presenter: Berger, Michael

Faculty Sponsor: Pullen, Nicholas

#### **Abstract:**

Myeloid-derived suppressor cells (MDSC) are a heterogeneous population of immature myeloid cells. However, unlike the mature immune cells of the same lineage, MDSC have demonstrated the capacity to suppress the immune system. In previous studies, it has been demonstrated that MDSC accumulate in the tumor microenvironment (TME) of cancer patients. This makes MDSC a potent therapeutic target. A protein that aids in the protection of gastrointestinal mucosa called trefoil factor two (TFF2) activates signal cascades, although only recently has the link between TFF2 and the chemokine receptor CXCR4 been studied. The aim of this project was to characterize this signal-receptor pairing in vitro. Our MDSC populations were grown by culturing bone-marrow derived hematopoietic stem cells in a mix of 4T1 cancer cell medium and standard complete RPMI 1640. Flow cytometry was used successfully to establish the presence of CXCR4 on the cell surface of MDSC. To investigate CXCR4's capacity to bind to TFF2, co-immunoprecipitation experiments were done on MDSC cultures treated with recombinant TFF2. These experiments yielded positive results, leading us to further study of the link between TFF2 and CXCR4. Given that CXCR4 is a chemokine, the next step in evaluating its interaction with TFF2 is measuring migration in MDSC treated with TFF2. To do this, we will be performing trans-well migration assays with MDSC and the TFF2-producing 4T1 cancer cell line. These assays will be done with and without an anti-TFF2 antibody to block its potential effects. If, as we predict, migration is reduced in the TFF2 deficient trans-well, then we can conclude that tumor-secreted TFF2 aids in the migration of myeloid cells from the bone marrow to the TME.